Doan, C. A.

May 22, 1936.

Dear Doctor Doan:

I was so glad to get your letter yesterday but so deeply sorry over the death of Mrs. Doan's Mother. How dreadfully sad for her father. I cannot imagine his getting along without her. What a hard winter you two have had!

I had a talk the other day with Doctor Alan Gregg in which he gave you very high praise for the way you have built up a fine organization in surroundings somewhat unsympathetic toward research. He felt that you had shown a very high power of organization.

I was exceedingly interested in what you said about your own research. I believe that there is a lot of interesting work to be done on monocytic leucemia. I have been trying hard to get a chance to see those slides of monocytic leucemia from the case that Doctor Mercer had. So far I have not yet succeeded. I will let you know if I ever do see them.

I want to call your attention to two articles. One, by Doctor M. H. Knisely, working in Bensley's laboratory, is on the circulation in the spleen. The reference is Anatomical Record, 1936, 65, 23. He has devised a way for watching circulation in the living spleen. He finds that the sinuses are really closed vessels, that they fill with blood, sphincters contract at either end, and all the fluid passes out in the pulp spaces but none of the corpuscles. These vessels may remain closed for a few minutes or a few hours. Eventually, when they open, the corpuscles are matted together and very dry but they eventually separate again. He cannot quite see how the fluid gets back because it is so colorless. I am going to ask him if he cannot put a dye into the plasma and see it go back. He sees an occasional corpuscle in the pulp spaces and finds that they snap back into the vessels. You will be interested in his paper which I think a very beautiful piece of work. I am going to write him and ask if by chance I could see the demonstration on my way through Chicago this year.

I am so sorry that I cannot stop to see you. My sister is with me and we are going through to Detroit to Mr. Swen's wedding and then directly on to Chicago.

The other paper I want to call to your attention is a Monograph by Helge Sjövall, published from the Institute for Microscopical Anatomy of the University of Lund, 1936. It is on lymphocytosis and I think that Doctor Wiseman should read it. He has a most extraordinary lymphocyte count, finding that in the normal neutrophilic leucocytes run about 1,800 per cu.mm. and lymphocytes

6,000 per cu.mm., and that after bleeding the lymphocytes suddenly drop to 4,000! What he has been counting for lymphocytes I just cannot imagine, unless his technique damages leucocytes. If you did not receive this reprint from him, I am inclined to loan you my copy for the summer if you think you might be interested in it. I should like to have it back in the fall. His whole theories which seem to me somewhat fantastic are on lymphocytes.

Concerning Doctor Wiseman's concept of the reciprocal relation between lymphocytes and leucocytes I do not feel quite sure. The one point that is clear enough is that when there develops a tremendous drive for the myeloid series, as in myelogenous leucemia, there is ultimately a lowering of the number of leucocytes. Is that not so? Concerning the other work, I think it perfectly wonderful -- a great clarification of the whole subject of lymphocytic disorders.

I think I wrote you sometime ago that we were interested in Hodgkin's. We think we have found out this, but we won't make any definite statement until we have seen more cases, that we can discriminate in the supravital between Hodgkin's and the so-called reticular cell sarcoma in this way: Both of them have exactly the same damage to the nucleus and the damage is far greater than any damage in the cytoplasm, but when the cases are truly Hodgkin's these cells are really monocytes of the Forkner type with the rosettes of fine neutral red bodies staining well. When, on the other hand, the case is really reticular cell sarcoma, it is the more primitive cell that is involved and there is no staining with neutral red at all, so my hunch is that these two represent the same disease, only in one case the cell involved is much more primitive and is really the forerunner of the monocyte rather than the monocyte itself and in this case the process involves a much greater multiplication of the cell involved. This is all very tentative, for we have not gone far.

Cordially yours,

Florence R. Sabin.

Doctor Charles A. Doan,
Department of Medical and
Surgical Research,
Ohio State University,
Columbus, Ohio.